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=> PTX3 and myocardial infarction
L1            0 FILE AGRICOLA
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L9    ANSWER 1 OF 6  PASCAL  COPYRIGHT 2005 INIST-CNRS. ALL RIGHTS RESERVED. on
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ACCESSION NUMBER:         2005-0278341   PASCAL
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reserved.
TITLE (IN ENGLISH): Effect of the toll-like receptor 4 (TLR-4) variants on intima-media thickness and monocyte-derived macrophage response to LPS
AUTHOR: NORATA G. D.; GARLASCHELLI K.; ONGARI M.; RASELLI S.; GRIGORE L.; BENVENUTO F.; MAGGI F. M.; CATAPANO A. L.
CORPORATE SOURCE: Department of Pharmacological Sciences, University of Milan, Milan, Italy; Center for the Study of Atherosclerosis, Ospedale Bassini, Cinisello Balsamo, Italy
SOURCE: Journal of internal medicine, (2005), 258(1), 21-27, 33 refs.
ISSN: 0954-6820
DOCUMENT TYPE: Journal
BIBLIOGRAPHIC LEVEL: Analytic
COUNTRY: United Kingdom
LANGUAGE: English
AVAILABILITY: INIST-893A, 354000138097940030
AN 2005-0278341 PASCAL
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AB Objectives. Toll-like receptor 4 (TLR-4) is believed to contribute to the initiation and progression of atherosclerosis. The association of the D299G polymorphism of the TLR-4 gene with the progression of coronary and carotid atherosclerosis, risk of cardiovascular events and **myocardial infarction** is controversial. We have investigated whether the presence of the D299G polymorphism and the co-segregated T399I polymorphism affects the intima-media thickness (IMT) in the general population. Subjects. The PLIC study population (n = 1256) was genotyped for the D299G and the T399I polymorphisms. Results. The presence of both the D299G and T399I alleles was observed in the 13.0% of the population, carriers of the T399I alone were 1.8% and of the D299G alone were 0.9%. No difference in IMT was detected within the carriers of the D299G and T399I alleles and the wild-type subjects in the PLIC population. Furthermore, we investigated whether monocyte from D299G to T399I subjects present a defective response to CD40, interleukin (IL)-6, monocyte chemotactic protein (MCP)-1, cyclooxygenase (COX)-2 and **PTX3** expression induced by lipopolysaccharide (LPS). When the monocyte-derived macrophages of these subjects were challenged with LPS (1 μg mL.sup.-.sup.1), no impact of the polymorphisms on the induction of CD40, MCP-1 and **PTX3** was observed. Only IL-6 and COX-2 induction by LPS resulted reduced in the D299G/ T399I carriers. Conclusion. The presence of the D299G and T399I polymorphisms of the TLR-4 gene does not play a major role on the progression of carotid atherosclerosis. Macrophages from the subjects carrying the polymorphisms show an impaired response to LPS limited only to a IL-6 and COX-2.

L9 ANSWER 2 OF 6 PASCAL COPYRIGHT 2005 INIST-CNRS. ALL RIGHTS RESERVED. on STN
ACCESSION NUMBER: 2005-0173158 PASCAL
COPYRIGHT NOTICE: Copyright .COPYRGT. 2005 INIST-CNRS. All rights reserved.
TITLE (IN ENGLISH): Prognostic significance of the long pentraxin **PTX3** in acute **myocardial infarction**
AUTHOR: LATINI Roberto; MAGGIONI Aldo P.; PERI Giuseppe; GONZINI Lucio; LUCCI Donata; MOCARELLI Paolo; VAGO Luca; PASQUALINI Fabio; SIGNORINI Stefano; SOLDATESCHI Dario; TARLI Lorenzo; SCHWEIGER Carlo; FRESCO Claudio; CECERE Rossana; TOGNONI Gianni; MANTOVANI Alberto
CORPORATE SOURCE: Mario Negri Institute for Pharmacological Research, Milan, Italy; ANMCO Research Centre, Firenze, Italy; Milano-Bicocca University, Department of Laboratory Medicine-Desio Hospital, Desio, Milan, Italy;

University of Milan, L. Sacco Hospital, Milan, Italy; Diesse-Diagnostica Senese SpA., Monteriggioni, Siena, Italy; Department of Cardiac Rehabilitation, Passirana Hospital, Rho-Milano, Italy; Department of Cardiology, S. Maria della Misericordia Hospital, Udine, Italy; Consorzio Mario Negri Sud, S. Maria Imbaro, Chieti, Italy; Institutes of Pathology and General Pathology, Milan, Italy
Lipid Assessment Trial Italian Network (LATIN) Investigators, Italy

SOURCE: Circulation : (New York, N.Y.), (2004), 110(16), 2349-2354, 40 refs.
ISSN: 0009-7322 CODEN: CIRCAZ
DOCUMENT TYPE: Journal
BIBLIOGRAPHIC LEVEL: Analytic
COUNTRY: United States
LANGUAGE: English
AVAILABILITY: INIST-5907, 354000125014420140
AN 2005-0173158 PASCAL

AB Background-Inflammation has a pathogenetic role in acute **myocardial infarction** (MI). Pentraxin-3 (**PTX3**), a long pentraxin produced in response to inflammatory stimuli and highly expressed in the heart, was shown to peak in plasma 7 hours after MI. The aim of this study was to assess the prognostic value of **PTX3** in MI compared with the best-known and clinically relevant biological markers. Methods and Results-In 724 patients with MI and ST elevation, **PTX3**, C-reactive protein (CRP), creatine kinase (CK), troponin T (TnT), and N-terminal pro-brain natriuretic peptide (NT-proBNP) were assayed at entry, a median of 3 hours, and the following morning, a median of 22 hours from symptom onset. With respect to outcome events occurring over 3 months after the index event, median **PTX3** values were 7.08 ng/mL in event-free patients, 16.12 ng/mL in patients who died, 9.12 ng/mL in patients with nonfatal heart failure, and 6.88 ng/mL in patients with nonfatal residual ischemia (overall P<0.0001). Multivariate analysis including CRP, CK, TnT, and NT-proBNP showed that only age >=70 years (OR, 2.11; 95% CI, 1.04 to 4.31), Killip class >1 at entry (OR, 2.20; 95% CI, 1.14 to 4.25), and **PTX3** (>10.73 ng/mL) (OR, 3.55; 95% CI, 1.43 to 8.83) independently predicted 3-month mortality. Biomarkers predicting the combined end point of death and heart failure in survivors were the highest tertile of **PTX3** and of NT-proBNP and a CK ratio >6. Conclusions-In a representative contemporary sample of patients with MI with ST elevation, the acute-phase protein **PTX3** but not the liver-derived short pentraxin CRP or other cardiac biomarkers (NT-proBNP, TnT, CK) predicted 3-month mortality after adjustment for major risk factors and other acute-phase prognostic markers.

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ACCESSION NUMBER: 2004-0599266 PASCAL

TITLE (IN ENGLISH): Modified atherogenic lipoproteins induce expression of pentraxin-3 by human vascular smooth muscle cells
AUTHOR: KLOUCHE Mariam; PERI Giuseppe; KNABBE Cornelius; ECKSTEIN Hanns-Henning; SCHMID Franz-Xaver; SCHMITZ Gerd; MANTOVANI Alberto
CORPORATE SOURCE: Institute of Clinical Chemistry and Laboratory Medicine, University of Regensburg, Franz-Josef-Strauβ Allee 11, 93053 Regensburg, Germany, Federal Republic of; Istituto di Ricerche Farmacologiche Mario Negri, Milan, Italy; Istituto di

Patologia Generale, Universita di Milano, Milan, Italy; Robert-Bosch-Hospital, Stuttgart, Germany, Federal Republic of; Department of Vascular Surgery, Ludwigsburg, Germany, Federal Republic of; Department of Vascular and Thoracic Surgery, University of Regensburg, Regensburg, Germany, Federal Republic of

SOURCE: Atherosclerosis, (2004), 175(2), 221-228, 38 refs. ISSN: 0021-9150

DOCUMENT TYPE: Journal
BIBLIOGRAPHIC LEVEL: Analytic
COUNTRY: Netherlands
LANGUAGE: English
AVAILABILITY: INIST-1713, 354000113823640040
AN 2004-0599266 PASCAL

AB Inflammation is a critical contributing factor to the development and the progression of atherosclerosis. Recently, the acute-phase protein pentraxin-3 (**PTX3**), which has C-terminal sequence homology with the classic pentraxin C-reactive protein (CRP), was described to be increased in patients with **myocardial infarction**. In this study, we have investigated the capacity of human primary vascular smooth muscle cells (VSMC), derived from arterial specimens of ten different patients, to express **PTX3** after incubation with atherogenic lipoproteins. Enzymatically degraded LDL (E-LDL), which is present in human early lesions, mediated a rapid cholesterol loading and foam cell transformation of primary VSMC, which was paralleled by a marked dose- and time-dependent expression of **PTX3** mRNA and release of the acute-phase protein. Expression of **PTX3** mRNA was delayed and remained almost undetectable for up to 6h of incubation with E-LDL. However, during extended exposure to E-LDL for more than 24 h, **PTX3** mRNA expression increased by more than 15-fold in VSMC foam cells, which was reflected by a concomitant release of up to 211 ng/ml **PTX3** protein. We provide evidence for marked expression of **PTX3** by VSMC induced by degraded lipoproteins, which may lead to an in situ vascular acute-phase reaction, contributing to the inflammatory pathogenesis of atherosclerosis.

L9 ANSWER 4 OF 6 LIFESCI COPYRIGHT 2005 CSA on STN
ACCESSION NUMBER: 2005:14101 LIFESCI
TITLE: The long pentraxin **PTX3** up-regulates tissue factor in activated monocytes: another link between inflammation and clotting activation
AUTHOR: Napoleone, E.; Di Santo, A.; Peri, G.; Mantovani, A.; de Gaetano, G.; Donati, M.B.; Lorenzet, R.
CORPORATE SOURCE: "Antonio Taticchi" Unit for Atherosclerosis and Thrombosis, Istituto Ricerche Farmacologiche Mario Negri, Consorzio Mario Negri Sud, Via Nazionale, 66030 S. Maria Imbaro, Italy; E-mail: lorenzet@negrisud.it
SOURCE: Journal of Leukocyte Biology [J. Leukocyte Biol.], (20040700) vol. 76, no. 1, pp. 203-209. ISSN: 0741-5400.
DOCUMENT TYPE: Journal
FILE SEGMENT: F
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Pentraxin-3 (**PTX3**), an acute-phase protein that belongs to the family of the PTXs, is found elevated in septic shock and increased in patients with acute **myocardial infarction**. As tissue factor (TF) plays a key role in thrombosis and inflammation associated with atherosclerosis and as we have recently reported that **PTX3** increases TF synthesis in endothelial cells, we tested whether **PTX3** could modulate TF expression in monocytes. Monocytes from peripheral blood of healthy donors were incubated with highly purified

PTX3 with or without lipopolysaccharide (LPS). Cells were then disrupted, and procoagulant activity was assessed by a one-stage clotting time. **PTX3** enhanced TF activity and antigen from LPS-stimulated monocytes in a dose-dependent way. The effect was specific, as other PTXs, such as C-reactive protein and serum amyloid P component, were ineffective. Moreover, the increase in activity was specific for LPS, as in the presence of other TF-inducing agents such as interleukin-1 beta and tumor necrosis factor alpha , **PTX3** was not effective. The increase in TF activity requires mRNA synthesis, as assessed by polymerase chain reaction. The mechanism by which **PTX3** modulates TF synthesis resides in an enhanced I Kappa B, alpha phosphorylation and degradation and increased migration of the transacting factor c-Rel/p65 into the nucleus, as determined by Western blot and electro-mobility shift assay. These results show that **PTX3** is an enhancer of the expression of TF by mononuclear cells. In the area of vascular injury, during the inflammatory response, cell-mediated fibrin deposition takes place. **PTX3** increases TF expression, thus potentially playing a role in thrombogenesis and wound healing.

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ACCESSION NUMBER: 2002-0366555 PASCAL
COPYRIGHT NOTICE: Copyright .COPYRGT. 2002 INIST-CNRS. All rights reserved.
TITLE (IN ENGLISH): Long pentraxin **PTX3** upregulates tissue factor expression in human endothelial cells: A novel link between vascular inflammation and clotting activation
AUTHOR: NAPOLEONE Emanuela; DI SANTO Angelomaria; BASTONE Antonio; PERI Giuseppe; MANTOVANI Alberto; DE GAETANO Giovanni; DONATI Maria Benedetta; LORENZET Roberto
CORPORATE SOURCE: "Antonio Taticchi" Unit for Atherosclerosis and Thrombosis, Department of Vascular Medicine and Pharmacology, Istituto Ricerche Farmacologiche Mario Negri, Consorzio Mario Negri Sud, S. Maria Imbaro, Milano, Italy; Department of Immunology and Cell Biology, Istituto di Ricerche Farmacologiche Mario Negri, Milano, Italy; Istituto di Patologia Generale, University of Milano, Milano, Italy
SOURCE: Arteriosclerosis, thrombosis, and vascular biology, (2002), 22(5), 782-787, 36 refs.
ISSN: 1079-5642 CODEN: ATVBFA
DOCUMENT TYPE: Journal
BIBLIOGRAPHIC LEVEL: Analytic
COUNTRY: United States
LANGUAGE: English
AVAILABILITY: INIST-19104, 354000100710260110
AN 2002-0366555 PASCAL
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AB Inflammation is a major contributing factor to atherosclerotic plaque development and ischemic heart disease. **PTX3** is a long pentraxin that was recently found to be increased in patients with acute **myocardial infarction**. Because tissue factor (TF), the in vivo trigger of blood coagulation, plays a dominant role in thrombus formation after plaque rupture, we tested the possibility that **PTX3** could modulate TF expression. Human umbilical vein endothelial cells, incubated with endotoxin (lipopolysaccharide) or the inflammatory cytokines interleukin-1β and tumor necrosis factor-a, expressed TF. The presence of **PTX3** increased TF activity and antigen severalfold in a dose-dependent fashion. **PTX3** exerted its effect at the transcription level, inasmuch as the increased levels of TF mRNA, mediated by the stimuli, were enhanced in its presence. The increase in mRNA determined by **PTX3** originated from an enhanced

nuclear binding activity of the transacting factor c-Rel/p65, which was mediated by the agonists and measured by electrophoretic mobility shift assay. The mechanism underlying the increased c-Rel/p65 activity resided in an enhanced degradation of the c-Rel/p65 inhibitory protein IκBα. In the area of vascular injury, during the inflammatory response, cell-mediated fibrin deposition takes place. Our results suggest that **PTX3**, by increasing TF expression, potentially plays a role in thrombogenesis and ischemic vascular disease.

L9 ANSWER 6 OF 6 PASCAL COPYRIGHT 2005 INIST-CNRS. ALL RIGHTS RESERVED. on STN

ACCESSION NUMBER: 2000-0431527 PASCAL

TITLE (IN ENGLISH): **PTX3**, a prototypical long pentraxin, is an early indicator of acute **myocardial infarction** in humans

AUTHOR: PERI G.; INTRONA M.; CORRADI D.; IACUITTI G.; SIGNORINI S.; AVANZINI F.; PIZZETTI F.; MAGGIONI A. P.; MOCCETTI T.; METRA M.; CAS L. D.; GHEZZI P.; SIPE J. D.; RE G.; OLIVETTI G.; MANTOVANI A.; LATINI R.

CORPORATE SOURCE: Istituto di Ricerche Farmacologiche Mario Negri, Milan, Italy; Department of Pathology, University of Parma, Italy; Division of Cardiology of Desio, Italy; Divisions of Cardiology of Seriate, Italy; Division of Cardiology of, Casale Monferrato, Italy; Division of Cardiology of Lugano, Lithuania; Division of Cardiology of Brescia, Italy; Department of Biochemistry, Boston University, Boston, Mass, United States; Clinical Chemistry Laboratory, Legnano Hospital, Italy; Department of Biotechnology, Section of General Pathology, University of Brescia, Italy

SOURCE: Circulation : (New York, N.Y.), (2000), 102(6), 636-641, 31 refs.
ISSN: 0009-7322 CODEN: CIRCAZ

DOCUMENT TYPE: Journal
BIBLIOGRAPHIC LEVEL: Analytic
COUNTRY: United States
LANGUAGE: English
AVAILABILITY: INIST-5907, 354000090939240080

AN 2000-0431527 PASCAL

AB Background-Inflammation is an important component of ischemic heart disease. **PTX3** is a long pentraxin whose expression is induced by cytokines in endothelial cells, mononuclear phagocytes, and myocardium. The possibility that **PTX3** is altered in patients with acute **myocardial infarction** (AMI) has not yet been tested. Methods and Results-Blood samples were collected from 37 patients admitted to the coronary care unit (CCU) with symptoms of AMI. **PTX3** plasma concentrations, as measured by ELISA, higher than the mean+2 SD of age-matched controls (2.01 ng/mL) were found in 27 patients within the first 24 hours of CCU admission. **PTX3** peaked at 7.5 hours after CCU admission, and mean peak concentration was 6.94±11.26 ng/mL. Plasma concentrations of **PTX3** returned to normal in all but 3 patients at hospital discharge and were unrelated to AMI site or extent, Killip class at entry, hours from symptom onset, and thrombolysis. C-reactive protein peaked in plasma at 24 hours after CCU admission, much later than **PTX3** ($P<0.001$). Patients >64 years old and women had significantly higher **PTX3** concentrations at 24 hours ($P<0.05$). **PTX3** was detected by immunohistochemistry in normal but not in necrotic myocytes. Conclusions-**PTX3** is present in the intact myocardium, increases in the blood of patients with AMI, and disappears from damaged myocytes. We suggest that **PTX3**

is an early indicator of myocyte irreversible injury in ischemic cardiomyopathy.

Ref #	Hits	Search Query	DBs	Default Operator	Plurals	Time Stamp
L1	8	PTx3 same myocard$	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/12/07 11:26
L2	8299	(435/7.1,7.2,7.92).CCLS.	USPAT; EPO	OR	OFF	2005/12/07 11:28
L3	16158	(435/6;91.2).CCLS.	USPAT; EPO	OR	OFF	2005/12/07 11:28
L4	0	l1 and l2	USPAT; EPO	OR	OFF	2005/12/07 11:28
L5	2	l1 and l3	USPAT; EPO	OR	OFF	2005/12/07 11:28